

The effect of blunt abdominal trauma on appendix vermiformis

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Objectives: Trauma and appendicitis are the most common conditions of childhood for which surgical consultation is sought in emergency departments. Occasionally, appendicitis and trauma exist together, which causes an interesting debate whether trauma has led to appendicitis. We aimed to evaluate our patients with traumatic appendicitis and to discuss their properties in the light of the literature.

Methods: We retrospectively reviewed the charts of children of blunt abdominal trauma accompanied by appendicitis.

Results: Of 29 cases of blunt abdominal trauma that had required surgical exploration, five were found to have gross findings of acute appendicitis and underwent appendicectomy. Appendicitis was confirmed histopathologically.

Conclusion: It should be kept in mind that children managed for severe blunt abdominal trauma may develop appendicitis. If clinical outlook suggests appendicitis in cases conservatively managed for blunt abdominal trauma, physical examinations, abdominal ultrasonography and/or abdominal computed tomography should be repeated for diagnosis of traumatic appendicitis. This approach will help to protect the patients against the complications of appendicitis that are likely to develop.

Trauma and appendicitis are the two most common conditions of childhood for which surgical consultation is made in the emergency department (ED).¹ The pathological process in appendicitis generally starts with obstruction of the appendiceal lumen and may progress to peritonitis and development of intraabdominal abscess via appendiceal inflammation and perforation. Trauma is a condition that causes multisystem injuries in childhood with both local and systemic effects. Occasionally, appendicitis and trauma coexist, and there is debate over the role of trauma in appendicitis.^{2,3} We aimed to evaluate our patients with traumatic appendicitis (TA) retrospectively and to discuss their properties in the light of relevant literature.

METHODS AND RESULTS

We retrospectively reviewed the charts of children with blunt abdominal trauma (BAT) accompanied by appendicitis, who were admitted to Aydın State Hospital and managed by the staff paediatric surgeon (MY) between August 1993 and April 1997 and the children managed by the same surgeon following his appointment to Adnan Menderes University Medical Center in Aydın between May 1997 and December 2002.

Case 1

A 5 year old boy was brought to the ED 4 hours after a rural motor vehicle accident. A wheel of a tractor had run over his abdomen. He complained of diffuse abdominal pain. Vital signs were as follows: blood pressure was 80/30 mmHg, heart rate 110 beats/min, and temperature 36.5°C. Laboratory investigations showed Hb 8.0 g/dl and white blood cell count (WBC) 18 700/mm³. He had a large contusion across his upper abdomen, and the abdomen was distended and diffusely tender. Abdominal ultrasonography showed multiple hepatic lacerations and large abdominal free fluid. On admission, intravenous fluid resuscitation, nasogastric suction and insertion of a Foley catheter were performed. In the ED, his blood pressure and haemoglobin continued to drop and abdominal signs worsened. Two hours after admission,

he was taken to the operating theatre. During laparotomy, multiple bleeding hepatic lacerations were located and sutured, and on exploration, he was found to have acute appendicitis. Appendicectomy was also performed and diagnosis of appendicitis was confirmed pathologically. The postoperative course was uneventful and he was discharged on postoperative day 15.

Case 2

An 8 year old boy was brought to the ED within 1 hour of a motor vehicle accident. A wheel of a minibus had run over his abdomen. There was an ecchymosis in the shape of a wheel on his abdominal wall. Vital signs were: blood pressure was 80/40 mmHg, heart rate 108 beats/min, and temperature 38.5°C. The initial laboratory results were as follows: Hb was 8.7 g/dl and WBC 19 500/mm³. On physical examination, he had diffuse abdominal pain and tenderness with mild pallor. Breath sounds were diminished on the right side. Right diaphragmatic irregularity on chest radiograph suggested rupture of the diaphragm. Abdominal ultrasonography showed a large hepatic laceration with large volume of abdominal free fluid and retroperitoneal haematoma. After massive fluid resuscitation, abdominal exploration was performed. At laparotomy, a right diaphragmatic rupture, a large laceration on the right lobe of the liver, and a right paracolic retroperitoneal haematoma were detected. On further exploration, acute appendicitis was encountered. The appendix was hyperoemic, oedematous, and thickened, and was removed. Appendicitis was confirmed pathologically. The postoperative course was uneventful and tube thoracostomy was discontinued on postoperative day 12. He was discharged on postoperative day 19.

Abbreviations: BAT, blunt abdominal trauma; CT, computed tomography; ED, emergency department; IAP, intra-abdominal pressure; TA, traumatic appendicitis; WBC, white blood cell count

Case 3

A 14 year old boy sustained BAT during a car crash. Within an hour of the accident, he was brought to the ED and hospitalised. He had ecchymoses on his right chest wall and right leg. Vital signs were: blood pressure was 100/60 mmHg; heart rate 72 beats/min, and temperature 36.8°C. The initial laboratory results were: Hb 12.5 g/dl and WBC 12 200/mm³. On physical examination, he had mild abdominal pain on the right upper quadrant but no tenderness. Breath sounds were diminished on the right side and breathing was painful, especially on the right side. Chest radiograph showed right lung contusion and costal fractures (ribs 9–12). There was an additional fracture on his right fibula. Abdominal ultrasonography showed a retroperitoneal haematoma on the right side. He was admitted to the intensive care unit. Fluid resuscitation and nasogastric suction were initiated. While under observation, his abdomen became distended, and abdominal pain increased and became diffused. On repeated abdominal examination, abdominal tenderness progressively worsened. Diffuse abdominal rebound tenderness was observed at the ninth hour postadmission. Nasogastric aspirate was bilious. WBC increased to 18 700 mm³ and fever of 38°C was observed in this period. Repeated ultrasonography revealed no additional information except a non-enlarging left retroperitoneal haematoma. He underwent surgery for acute abdomen and suspected intestinal necrosis. At exploration, a retroperitoneal haematoma was found, and the appendix was hyperaemic and inflamed. Appendicectomy was performed. Pathological examination confirmed acute appendicitis. Postoperative course was uneventful. He was discharged on postoperative day 15.

Case 4

A 9 year old boy sustained trauma follo fall from a height of 10 metres, and he arrived at our ED within an hour of the accident. Blood pressure was 80/50 mmHg; heart rate 86 beats/min and temperature 36.7°C. He had several superficial abrasions on his head and left chest. Physical examination revealed that he was confused and had mild respiratory distress. Breath sounds were diminished on the left side. On physical examination, he had mild abdominal tenderness. However, abdominal examination was not reliable because of head trauma. He was admitted to the intensive care unit. Fluid resuscitation and nasogastric suction were initiated. The laboratory test results were as follows: Hb was 11.2 g/dl, WBC was 17 700/mm³, and serum electrolytes and amylase were normal. Chest radiograph showed that he had left lung contusion, and CT confirmed this contusion. On abdominal radiograph, intraperitoneal subdiaphragmatic free air was observed. He had an additional fracture on his right fibula. On cranial CT, it was confirmed that he had left parietal fracture and diffuse brain oedema. Abdominal ultrasound and CT showed no additional intra-abdominal injury except free air, for which he underwent surgery. At abdominal exploration, there was no intestinal perforation observed but the appendix was found

to be hyperemic, oedematous, and inflamed appendix, and appendicectomy was performed. Pathological examination confirmed acute appendicitis without perforation. Postoperative course was uneventful. He was discharged on postoperative day 10.

Case 5

A 13 year old boy was brought to the ED 15 minutes after he had been involved in a motor-vehicle accident as a pedestrian. A wheel of a car had run over his abdomen and lower chest. He had several abrasions on his lower chest and abdominal wall, especially on the left side. His blood pressure was 110/60 mmHg, heart rate 112 beats/min, and temperature 37.1°C. The initial laboratory results showed Hb 10.1 g/dl and WBC 19 400/mm³. On physical examination, he had diffuse abdominal pain and tenderness. Breath sounds were normal. Left diaphragmatic elevation and irregularity on chest radiograph suggested diaphragmatic rupture. Thoracic and abdominal CT showed splenic laceration with a large volume of abdominal free fluid, pneumomediastinum, left hemidiaphragmatic rupture, rupture of the left ureteropelvic junction, and urinoma. After massive fluid resuscitation, laparotomy was performed at the third hour postinjury. Left diaphragmatic rupture, major splenic laceration and rupture of the left ureteropelvic junction were diagnosed intraoperatively. The left hemidiaphragm was repaired and the spleen was preserved with primary horizontal mattress suturing. Pyeloplasty was performed for the left ureteropelvic junction rupture. In addition to these pathologies, acute appendicitis was detected, and the appendix, which was hyperaemic, oedematous, and thickened, was removed. Appendicitis was confirmed pathologically. Postoperative course was uneventful and he was discharged on postoperative day 21.

Review

The mechanisms of trauma, associated injuries, and duration of preoperative period following trauma, physical findings and laboratory results of the cases with TA are shown in table 1. None of the patients diagnosed with TA had any symptoms in their histories suggesting acute abdomen before the traumatic insult. Abdominal CT was performed in only cases 4 and 5. The scan was not dedicated to reveal appendicitis. Neither oral nor rectal contrasts were used, and the appendix could not be visualised. On histopathological examination, the full thickness of the appendix wall was inflamed, with some periappendicitis observed in all cases.

DISCUSSION

The most commonly encountered pathophysiological mechanism of appendicitis is obstruction of its lumen by miscellaneous factors such as inspissated stool, foreign bodies, and intestinal parasites. Endometriosis, leukaemia, and emotional stress make up the non-obstructive but rare aetiologies.² Mucosal and submucosal inflammation secondary to bacterial invasion of lymphoid tissue of the appendix

Table 1 Mechanism of trauma and associated injuries of patients with TA before the operation

Case	Mechanism of trauma	Injuries	Comp	TI	Ab pain	RT	Fever (°C)	WBC (/mm ³)	Urine analysis
1	MVA	HL	HS	6	Diffuse	+	36.5	18 700	20–25 erythrocytes
2	MVA	HL, DR, RH	HS	3	Diffuse	+	38.5	19 500	Normal
3	MVA	RH, fracture of the right fibula, costal fractures (9–12)		9	Diffuse	+	36.8	12 200	Normal
4	Fall	Head trauma, chest trauma, intraperitoneal free air		6	Diffuse	+	36.7	17 700	Abundant erythrocytes
5	MVA	SL, DR, rupture of the left ureteropelvic junction		3	Diffuse	+	37.1	19 400	Abundant erythrocytes

*TI, time interval: the interval between traumatic insult and operation time. Comp, complications; Ab, abdominal; RT, rebound tenderness; MVA, motor vehicle accident; RH, retroperitoneal haematoma; HL, hepatic laceration; HS, hypovolaemic shock; DR, diaphragmatic rupture; SL, splenic laceration; WBC, white blood cell.

has also been claimed to be a factor of non-obstructive aetiology for appendicitis. This inflammation may resolve spontaneously or may progress to appendicitis.⁴

BAT has been suggested to be another aetiological factor causing appendicitis.^{3,4} Several criteria have been postulated for an appendicitis case to have a traumatic aetiology: presence of TA, no abdominal pain prior to trauma, blunt but severe traumatic insult of short duration directly to the abdomen, and an interval of 6–48 hours until emergence of symptoms following trauma.^{4,5} We encountered 45 cases, 16 of which were children fulfilling the criteria of TA reported in the literature.^{2,3,5-9} Diagnosis of TA is not easy in a child as the trauma or its complications are themselves able to elicit pain. This may cause delays in both diagnosis and treatment.^{9,10} Clinical suspicion is of utmost importance in such patients.^{6,8}

The relationship between appendicitis and trauma is not yet clearly identified. There are reports that discuss the effect of direct trauma as an aetiology. Hennington *et al.* in their report on one adult and one paediatric patient noted that following BAT with a direct effect on the appendix, appendiceal oedema, inflammation, and/or hyperplasia of intrinsic lymphoid tissues had resulted in obstruction of the appendiceal lumen.³ Çiftçi *et al* reported a series of five children with TA following direct trauma, suggesting the same mechanism.² Oedema and haematoma of the caecal wall and the root of the appendix, revealing possible direct trauma to the appendix, was identified in our case 2 but he also had associated intra-abdominal injuries. Supporting our findings, Serour *et al* have claimed that direct appendiceal injury is generally coexistent with other intra-abdominal organ injuries, and that the appendix is very rarely affected by direct trauma as it is very mobile and its dimensions very small.⁴

The indirect role of BAT in formation of appendicitis is much more obscure. It is well known that intra-abdominal pressure (IAP) increases in varying degrees in every BAT case.^{11,12} The mechanism of this increase in pressure is due to the relatively diminishing size of the abdominal cavity caused

by post-traumatic conditions such as external traumatic compression,¹³ intraperitoneal bleeding and/or retroperitoneal haematoma,^{12,14} acute gastric dilatation, and intraperitoneal free air occupying space within the abdominal cavity.¹² Increased IAP is a condition that may greatly interfere with visceral circulation. Engum *et al.* has discussed a technique to measure IAP for prevention of its complications. This technique depends on changes in the gastric mucosal blood flow, as the luminal surface of the bowel mucosa is suggested to be at a particular risk for ischaemia during the hypoperfusion observed in increased IAP.¹⁵

Ramscook has discussed an indirect mechanism resulting in TA, triggered by a sudden increase of IAP during trauma.⁵ According to this mechanism, a sudden increase in IAP may lead to an increased intracecal pressure followed by a rapid distention of the appendix. This may cause abrasion of the appendiceal mucosa together with a decrease in the appendiceal blood flow. He suggested that mucosal injury and decreased blood flow might result in appendicitis. In our four cases, there were findings that suggested a nonobstructive mechanism triggered by an increase in IAP via an indirect effect of trauma, but in our case 2, TA might have been a result of indirect and direct effects of trauma.

In our opinion, there is a multifactor complex mechanism in formation of TA. Visceral oedema indirectly contributes to the rise in IAP, and IAP may result from vigorous fluid administration during resuscitation.¹⁶ Furthermore, hypoperfusion as a complication of major haemorrhage may be followed by reperfusion injury, which may lead to visceral oedema.^{12,17} Oedema formation relatively diminishes the size of the abdominal cavity and increases IAP. A vicious cycle forms between increased IAP and visceral oedema (fig 1). It has been reported that in cases with normally maintained cardiac output and blood pressure, a rise in IAP of even <10 mm Hg may produce a significant decrease in visceral arterial blood flow, which is followed by oedema of the bowel wall.¹² The mechanism that Ramscook described might have played a role in our four cases, but we suggest that visceral oedema of the appendix may also contribute to

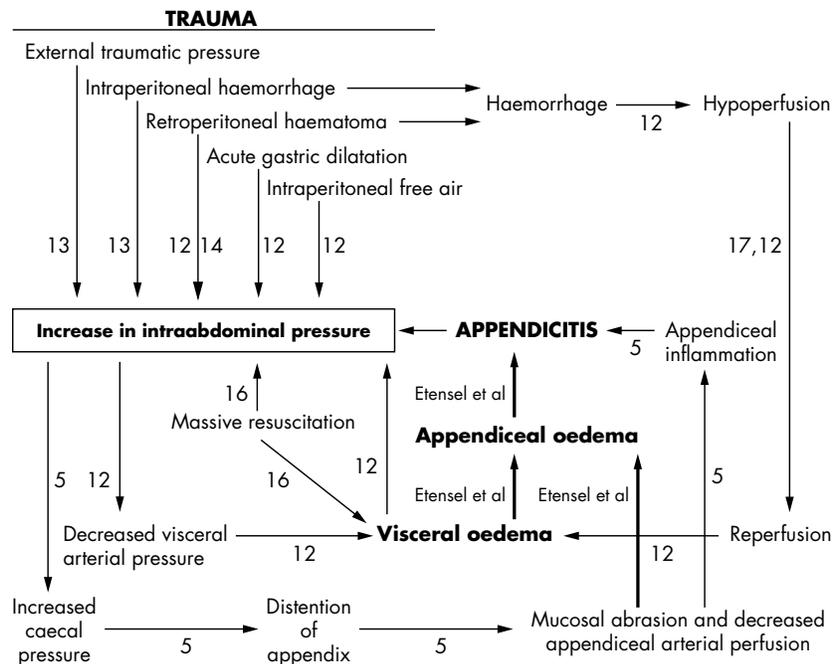


Figure 1 Pathophysiological mechanisms of traumatic appendicitis cited in the literature (numbered arrows) and our additional suggestion (Etensel *et al*).

formation of TA. The factors leading to visceral oedema were presence of hypovolaemic shock and use of massive fluid resuscitation for stabilisation in our case 2, and decrease in visceral arterial blood flow caused by increased IAP in the other two cases. Mucosal abrasion and decrease in appendiceal blood flow leading to increased oedema formation of the appendix may be other contributing factors in formation of TA.

High energy trauma may have shortened the interval from traumatic insult to surgery in our two cases of TA (cases 2 and 5). High energy traumas cause higher external traumatic compression and need aggressive fluid resuscitation. Furthermore, the blood loss-hypoperfusion-reperfusion chain is faster and more severe. This may lead to rapid development of severe visceral and appendiceal oedema, with resultant rapid development of inflammation.

In conclusion, appendicitis may follow trauma by several reasons. In children managed for severe trauma, development of TA should be also considered. If clinical progress suggests appendicitis in conservatively managed BAT, physical examinations, abdominal ultrasonography and/or abdominal CT should be repeated for diagnosis of TA. This approach will most probably prevent potential complications of appendicitis.

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